

## Novel genetic finding offers new avenue for future Crohn's disease treatment

## July 9 2009

Researchers from Case Western Reserve University School of Medicine identified a novel link between ITCH, a gene known to regulate inflammation in the body and NOD2, a gene which causes the majority of genetic Crohn's Disease diagnoses. ITCH, when malfunctioning, causes widespread inflammatory diseases, including inflammatory bowel disease, gastritis, uncontrolled skin inflammation, and pulmonary pneumonitis.

Derek Abbott, M.D., Ph.D., and his team of researchers found that ITCH also influences NOD2-induced inflammation. These findings, published in the August 11th issue of *Current Biology*, suggest a common pathophysiology exists between multiple inflammatory diseases. The unexpected finding of the interaction between these genes offers the possibility of a new <u>drug target</u>, which would be effective in treating <u>Crohn's disease</u> - a chronic disorder causing inflammation of the gastrointestinal tract.

Autoimmune and <u>inflammatory diseases</u> are striking an increasing portion of the population. They result from an overstimulation of the immune system by the infectious and environmental agents individuals face daily. Unfortunately, despite their increasing prevalence in the Western world and morbidity among younger patients, the pathophysiology of these enigmatic diseases is poorly understood and for this reason, treatment for these diseases is less-than-ideal.

This finding links two key signaling pathways to the pathophysiology of



diseases associated with ITCH and NOD2 and opens new avenues of pharmacologic pursuit to target these diseases. With an eye towards clinical applications, Dr. Abbott and his colleagues' next step is to determine if currently used pharmacologic agents can be useful in this model of inflammatory disease. They will do so using small molecule drug screening to identify potential drugs that target ITCH.

Of those diagnosed with Crohn's disease, 30 percent have the NOD2 mutation in their genes. For these individuals, this discovery opens up the possibility of individually-tailored treatments with better efficacy toward a particular patient's disease.

"This research is an excellent example of how scientific investments benefit the public with measureable gains. In this case, it led to unexpected insights and opened new fields of endeavor for pharmacological manipulation in this serious chronic disease," says Derek Abbott, M.D., Ph.D., assistant professor of pathology, Case Western Reserve University School of Medicine. "This sort of study will help uncover the pathologic mechanism of disease and ultimately lead to more rational and carefully measured treatment."

Source: Case Western Reserve University (<u>news</u> : <u>web</u>)

Citation: Novel genetic finding offers new avenue for future Crohn's disease treatment (2009, July 9) retrieved 24 April 2024 from <u>https://medicalxpress.com/news/2009-07-genetic-avenue-future-crohn-disease.html</u>

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